Breeding Table and Raisin Grapes with Increased Fruit Quality while Retaining Pierce's Disease Resistance

D.W. Ramming
Crop Diseases, Pests and
Genetics Research Unit
USDA/ARS
Parlier, CA
USA

M.A. Walker, A. Tenscher and
A.F. Krivanek
Dept. Viticulture and Enology
Univ. California, Davis
Davis, CA
USA

Keywords: grapevine, cultivars, PD resistance, breeding program

Abstract

The spread of Pierce's disease (PD) has greatly increased with the introduction of the glassy-winged sharpshooter into California. A collaborative breeding program to develop table and raisin grape cultivars resistant to PD was started in 2000. *V. arizonica/candicans* grape hybrids with PD resistance were hybridized with table and raisin grapes and the first generation screened in the greenhouse to determine resistance. The second generation was screened by molecular markers to identify resistant individuals. There was no difference between the first generation resistant and susceptible populations of D8909-15 x table grape for cluster weight, berry weight, and seed/trace weight. The cluster weight and berry size of the best resistant individuals were twice the size of the resistant parent. Aborted seeds, similar in size to the seedless parent, were also achieved in a resistant seedling. There was also no difference between first generation resistant and susceptible populations of F8909-08 x table grape selections for cluster size, berry size, and seed size. Resistant individuals with the largest berry size averaged 1.82 g compared to 3.6 to 14.4 g for the table grape parents. There was no difference between resistant and susceptible populations for berry size and seed/trace size in the second generation that resulted from backcrossing to table and raisin grapes. The mean berry size was 0.6 g larger and the mean seed/trace size decreased from 106 to 47 mg in the second generation. The second generation resistant individual with the largest berry averaged 4.9 g. Resistant individuals with undetectable seed traces, smaller than the seedless parents, were obtained in the second generation. This shows that fruit quality can be rapidly improved in the development of PD resistant grapes when efficient screening methods are used.

INTRODUCTION

Pierce's disease (PD) has existed in California since the late 1800s when it caused an epidemic in Anaheim. A number of vectors for PD already exist in California, causing its spread. The introduction of the glassy-winged sharpshooter to California in the 1990's increased the spread and damage caused by PD. Other vectors exist outside California and are always a threat. All of California's table and raisin grape cultivars grown commercially are susceptible to PD. A practical way to combat PD and its vectors is to develop PD-resistant varieties regardless of vector. PD resistance exists in a number of *Vitis* species and in the related genus, *Muscadinia*. Resistant cultivars have been developed by public (Dunstan, 1965; Loomis, 1958; Mortensen, 1977, 1983a, b; Olmo 1986; Overcash 1981, 1982) and private (Barrett, Bloodworth, Zehnder and others) breeding programs across the southeastern United States. These cultivars have high PD resistance, but relatively low fruit quality in comparison to *V. vinifera* table and raisin grape varieties grown in California. Rapid greenhouse screening techniques for detecting resistance to Xf and PD symptoms (Krivanek et al., 2005; Krivanek and Walker, 2005) have been developed and optimized. Embryo rescue techniques (Emershad et al., 1989; Emershad and Ramming, 1994) allow the recovery of very high frequencies of seedless progeny from seedless x seedless crosses. These techniques also allow the crossing of

Proc. IX th Intl. Conf. on Grape Genetics and Breeding
Eds.: E. Peterlunger et al.
Acta Hort. 827, ISHS 2009

high quality seedless table and raisin grapes with pollen from Xf resistant parents. Families between high quality table and raisin grapes hybridized with PD resistant grapes were analyzed to determine if fruit quality was increased as rapidly in resistant progeny as compared to susceptible progeny.

MATERIALS AND METHODS

Two sources of PD resistant germplasm, D8909-15 (*V. rupestris* x *V. arizonica*) and F8909-08 (*V. rupestris* x *V. arizonica/candicans*) were hybridized with advanced table grape selections having high fruit quality. Seeds were germinated from D8909-15 (*V. rupestris* x *V. arizonica*, seeded) x *V. vinifera* (seedless) (=Family 1) for plant recovery. Plants were recovered by embryo culture from *V. vinifera* (seedless) x F8909-08 (*V. rupestris* x *V. arizonica/candicans,* male flowered) (=Family 2). As soon as plants were large enough, duplicate plants were made for greenhouse testing of resistance to Xf. The greenhouse screening methodology is based on the pin-prick needle inoculation technique of Hopkins (1980, 1984). A 20 µl droplet of Xf suspension with about 10^8 colony forming units/ml is placed on a partially lignified stem just above the petiolar junction. A 25 gauge x 1.25 inch needle is then pushed through the droplet five times penetrating about one third of the width of the shoot. When this is done in a warm, sunny greenhouse, the suspension is quickly sucked into the stem by vascular pressure. PD symptoms are first seen about eight weeks after inoculation on susceptible cultivars like 'Chardonnay'. Plant symptoms and XF populations in the plants were characterized for Family 1 and 2 by previously reported methods (Krivanek et al., 2005; Krivanek and Walker, 2005) to determine resistant and susceptible plants. The Stag's Leap strain is currently being used in tests and previous results show no differences between it and the Santa Cruz or UCLA strain. Depending upon growth rate and weather, samples are taken after 12 to 16 weeks for ELISA evaluation. Male resistant selections from *V. vinifera* x F8909-08 were crossed on to seedless *V. vinifera* table and raisin grape selections (=family 2 BC1). Progeny from all three families were tested with a marker linked to *PdR1* (Krivanek et al., 2006) to identify PD resistant seedlings. As seedlings fruited in the field, fruit samples were taken to determine cluster size (n=3), berry size (n=50), and seed weight (all seeds from ten berry sample). These characteristics were used as measurements of fruit quality.

RESULTS AND DISCUSSION

There were no significant differences between the resistant and susceptible progeny for their average cluster weight, berry weight, and seed weight (Tables 1, 2, 3) in the three families observed. It is important to know that resistance is not linked to small clusters or berries or large seeds, so rapid progress can be made. The seed size ranged from small, aborted seeds to large seeds in Family 1 for both resistant and susceptible progeny (Fig. 1). A similar range in berry size existed (Fig. 2). The resistant seedlings had berries larger than the resistant parent. Forty percent of the resistant progeny in Family 1 were seedless, having aborted traces smaller than 35 mg. Seeds in Family 2 averaged almost twice the size of those in Family 1, however no seedless progeny were found for either resistant or susceptible individuals. The average berry size for resistant progeny in Family 1 and 2 was 0.6-0.8 g larger than the fruited resistant parent. The berry size for Family 2 averaged 0.2-0.5 g larger than Family 1, probably because only seeded individuals occurred. The resistant individual with the largest berry in Family 2 averaged 1.8 g. The seedless parents had berries averaging from 3.6 to 14.4 g and aborted seeds averaging from 8.9 to 28.5 mg. The average berry size for resistant progeny in Family 2 BC1 was 0.6 g larger than Family 2. The average seed weight was decreased from 106 mg to 47 mg and over half of the resistant seedlings had seedless fruit (Fig. 1). Resistant individuals with undetectable seed traces, smaller than the seedless parents, were obtained in the second generation Family 2 BC1. The resistant seedling with the largest berry averaged 4.9 g and was seeded. The resistant seedless seedling with the largest berries averaged 2.9 g with 18.0 mg aborted seeds.

CONCLUSIONS

There were no significant differences for cluster weight, berry weight, and seed weight between the resistant and susceptible progeny for the three families observed. Berry size increased significantly in the second generation Family 2 BC1 and a large number of seedless progeny were obtained. This shows that good progress can be made while keeping PD resistance.

Literature Cited

Dunstan, R.T. 1965. Hybridization of *Euvitis* X *Vitis rotundifolia*: backcrosses to muscadine. J. Am. Soc. Hort. Sci. 84:238-242.

Emershad, R.L., Ramming, D.W. and Serpe, M.D. 1989. In-ovulo embryo development and plant formation from stenospermic genotypes of *Vitis vinifera*. Am. J. Bot. 76:397-402.

Emershad, R.L. and Ramming, D.W. 1994. Effects of buffers and pH on *Vitis* somatic embryo proliferation and enlargement. Inter. Symp. Table Grape Production. Special Issue Am. Soc. Enology and Viticulture. p.219-222.

Hopkins, D.L. 1980. Use of pin-prick inoculation technique demonstrate variability in virulence of Pierce's disease bacterium. p.177-180. In: Proc. of VII Conf. Viruses of Grapevine (ICVG), 8-12 September 1980, Niagara Falls, Canada.

Hopkins, D.L. 1984. Variability of virulence in grapevine among isolates of the Pierce's disease bacterium. Phytopathology 74:1395-1398.

Krivanek, A.F. and Walker, M.A. 2005. *Vitis* resistance to Pierce's disease is characterized by differential *Xylella fastidiosa* populations in stems and leaves. Phytopathology 95:44-52.

Krivanek, A.F., Stevenson, J.F. and Walker, M.A. 2005. Development and comparison of symptom indices for quantifying grapevine resistance to Pierce's disease. Phytopathology 95:36-43.

Krivanek, A.F., Riaz, S. and Walker, M.A. 2006. Identification and molecular mapping of *PdR1*, a primary resistance gene to Pierce's disease in *Vitis*. Theor. Appl. Genet. 112:1125-1131.

Loomis, N.H. 1958. Performance of *Vitis* species in the south as an indication of their relative resistance to Pierce's disease. Plant Dis. Rep. 42:833-836.

Mortensen, J.A. and Stover, L.H. 1983a. 'Daytona' grape. HortScience 18:766-767.

Mortensen, J.A. and Stover, L.H. 1983b. 'Suwannee' and 'Conquistador' grapes. HortScience 18:767-769.

Mortensen, J.A., Stover, L.H. and Balerdi, C.F. 1977. Sources of resistance to Pierce's disease in *Vitis*. J. Am. Soc. Hort. Sci. 102:695-697.

Olmo, H.P. 1986. The potential role of (*vinifera* x *rotundifolia*) hybrids in grape variety improvement. Experimentia 42:921-926.

Overcash, J.P., Hedwood, Jr, C.P. and Stojanovic, B.J. 1981. 'Midsouth' and 'MissBlue' — two new bunch grape cultivars. Mississippi Ag and Forest. Exp Sta. Bulletin 6(18).

Overcash, J.P., Hedwood, Jr, C.P. and Stojanovic, B.J. 1982. 'Miss Blanc' — a new bunch grape cultivar. Mississippi Ag and Forest. Exp Sta. Bulletin 909.

Tables

Table 1. Average cluster weight, berry weight, and seed weight for resistant, intermediate, and susceptible progeny and parents of Family 1, 0023 = D8909-15 (*V. rupestris* x *V. arizonica*) (seeded) x B90-116 (*V. vinifera*).

	n	Avg. cluster wt. (g)	SD	n	Avg. berry wt. (g)	SD	n	Avg. seed wt. (mg)	SD
Resistant	7	20.4	11.6	12	1.22	0.42	12	47.0	23.2
Intermediate	7	17.7	15.5	9	1.20	0.43	9	45.8	25.7
Susceptible	44	18.9	16.2	68	1.18	0.48	67	53.9	20.4
D8909-15		16.3			0.63			66.8	
B90-116		383.8			9.1			15.5	

Table 2. Average cluster weight, berry weight, and seed weight for resistant, intermediate, and susceptible progeny of Family 2, *V. vinifera* x F8909-08 (*V. rupestris* x *V. arizonica/candicans*) (male flowered).

	n	Avg. cluster wt. (g)	SD	Avg. berry wt. (g)	SD	Avg. seed wt. (mg)	SD
Resistant	7	119.7	98.0	1.4	0.42	105.6	25.7
Intermediate	6	55.0	43.0	1.1	0.37	85.3	23.9
Susceptible	10	69.8	33.7	1.7	0.42	95.4	27.9

Table 3. Average cluster weight, berry weight, and seed weight for resistant, intermediate, and susceptible progeny in Family 2 BC1, *V. vinifera* x [*V. vinifera* x (*V. rupestris* x *V. arizonica/candicans*)].

	n	Avg. berry wt. (g)	SD	Avg. seed wt. (mg)	SD
Resistant	9	2.0	1.35	48.66	40.78
Intermediate	5	2.0	0.72	69.36	35.14
Susceptible	26	2.4	1.59	44.85	36.96

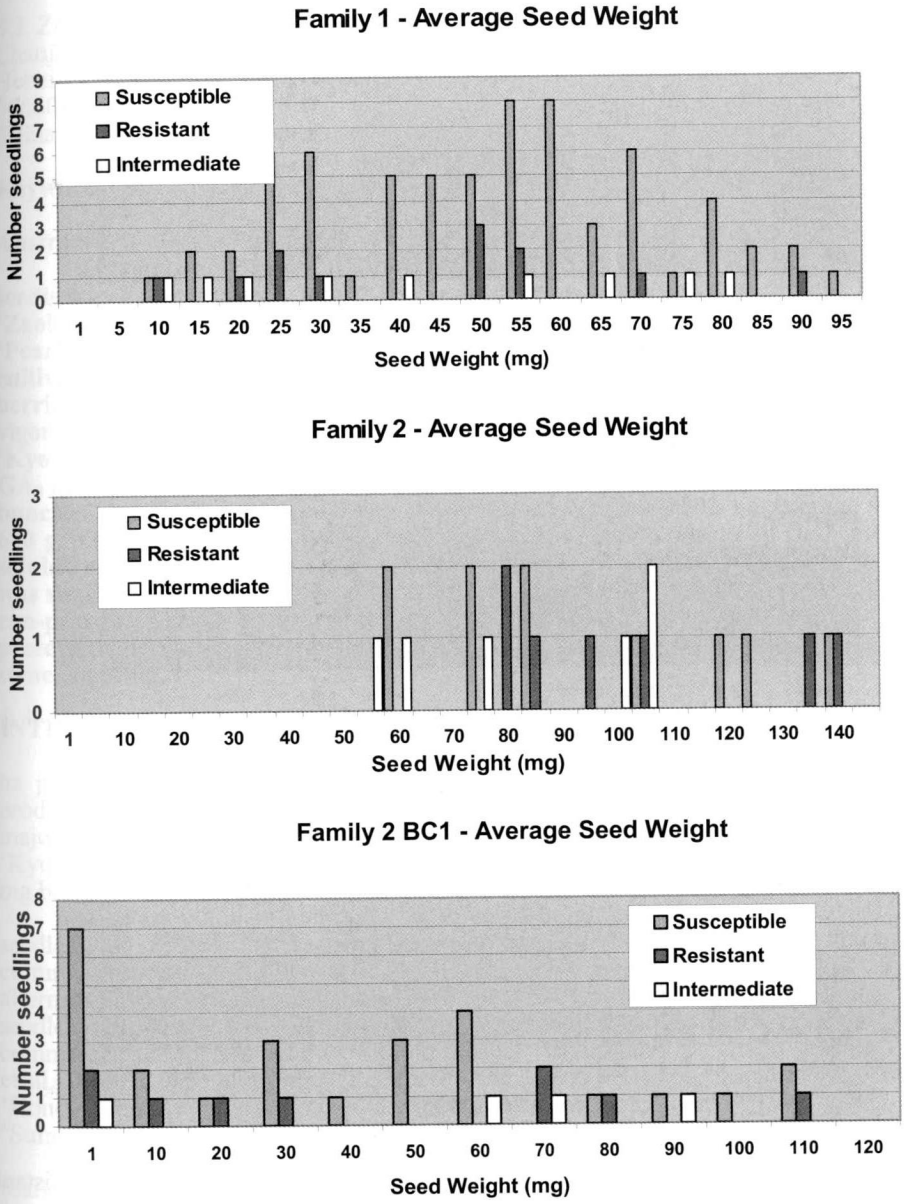

Fig. 1. Distribution of seed weight for the susceptible, intermediate, and resistant progeny in Family 1, Family 2, and Family 2 BC1.

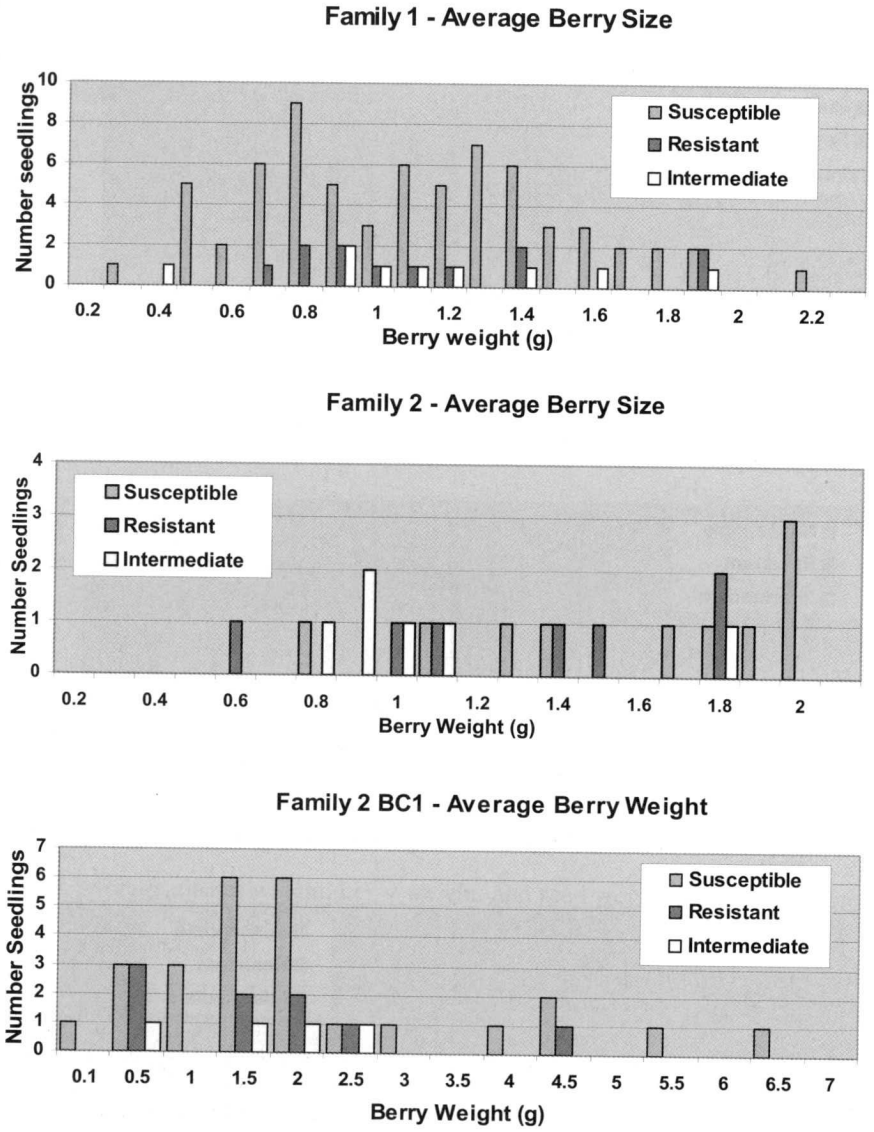

Fig. 2. Distribution of berry weight for the susceptible, intermediate, and resistant progeny in Family 1, Family 2, and Family 2 BC1.